

WEST Search History

DATE: Wednesday, October 17, 2007

<u>Hide?</u>	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L10	L9 and l8	0
<input type="checkbox"/>	L9	L7 and western blot	10
<input type="checkbox"/>	L8	L7 and cerebrospinal fluid	5
<input type="checkbox"/>	L7	L4 and @ay<1998	27
<input type="checkbox"/>	L6	L4 and ay<1998	150
<input type="checkbox"/>	L5	L4 and inhibit\$	141
<input type="checkbox"/>	L4	L3 and crosslinking	150
<input type="checkbox"/>	L3	L1 and redox	723
<input type="checkbox"/>	L2	L1 and redox metal	7
<input type="checkbox"/>	L1	amyloid beta or abeta or a beta	13121

END OF SEARCH HISTORY

FILE 'MEDLINE' ENTERED AT 11:10:55 ON 17 OCT 2007

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=> s amyloid beta or abeta
L1 50940 AMYLOID BETA OR ABETA

=> s redox reactive metal(p)crosslinking
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'METAL(P)CROSSLINKI'
L2 0 REDOX REACTIVE METAL(P) CROSSLINKING

=> s redox reactive metal and crosslinking
L3 0 REDOX REACTIVE METAL AND CROSSLINKING

=> s (redox reactive metal) and crosslinking
L4 0 (REDOX REACTIVE METAL) AND CROSSLINKING

=> s redox and crosslinking
L5 421 REDOX AND CROSSLINKING

=> s l1 and l5
L6 0 L1 AND L5

=> s l1 and metal
L7 1202 L1 AND METAL

=> s l1 and copper
L8 1092 L1 AND COPPER

=> s l1 and iron
L9 739 L1 AND IRON

=> s redox and l7
L10 198 REDOX AND L7

=> s l8 and l7
L11 587 L8 AND L7

=> s redox and l8
L12 164 REDOX AND L8

=> s l10 and l12
L13 118 L10 AND L12

=> s l9 and redox
L14 123 L9 AND REDOX

=> s l13 and l14
L15 71 L13 AND L14

Can # 10/643226
STN
10/17/07
As

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=> s l15 and cerebrospinal fluid
L16          6 L15 AND CEREBROSPINAL FLUID

=> s l15 and crosslinking
L17          0 L15 AND CROSSLINKING

=> dup rem l15
PROCESSING COMPLETED FOR L15
L18          35 DUP REM L15 (36 DUPLICATES REMOVED)

=> s l18 and py<1998
      2 FILES SEARCHED...
L19          0 L18 AND PY<1998

=> dup rem l8
PROCESSING IS APPROXIMATELY 95% COMPLETE FOR L8
PROCESSING COMPLETED FOR L8
L20          575 DUP REM L8 (517 DUPLICATES REMOVED)
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=> s l20 and py<1998
      1 FILES SEARCHED...
      5 FILES SEARCHED...
L21          23 L20 AND PY<1998
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=> disp l21 ibib abs 1-23
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L21  ANSWER 1 OF 23      MEDLINE on STN
ACCESSION NUMBER: 97477005      MEDLINE
DOCUMENT NUMBER: PubMed ID: 9337068
TITLE: Reactive oxygen species and Alzheimer's disease.
AUTHOR: Multhaup G; Ruppert T; Schlicksupp A; Hesse L; Behr D;
        Masters C L; Beyreuther K
CORPORATE SOURCE: ZMBH-Center for Molecular Biology Heidelberg, University of
        Heidelberg, Germany.. g.multhaup@mail.zmbh.uni-
        heidelberg.de
SOURCE: Biochemical pharmacology, (1997 Sep 1) Vol. 54,
        No. 5, pp. 533-9. Ref: 85
        Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
        General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 24 Dec 1997
        Last Updated on STN: 24 Dec 1997
        Entered Medline: 31 Oct 1997
```

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AB  Although a consensus that Alzheimer's disease (AD) is a single disease has
not been reached yet, the involvement of the amyloid precursor protein
(APP) and betaA4 (A beta) in the pathologic changes advances our
understanding of the underlying molecular alterations. Increasing
evidence implicates oxidative stress in the neurodegenerative process of
AD. This hypothesis is based on the toxicity of betaA4 in cell cultures,
and the findings that aggregation of betaA4 can be induced by
metal-catalyzed oxidation and that free oxygen radicals may be involved in
APP metabolism. Another neurological disorder, familial amyotrophic
lateral sclerosis (FALS), supports our view that AD and FALS may be linked
through a common mechanism. In FALS, SOD-Cu(I) complexes are affected by
hydrogen peroxide and free radicals are produced. In AD, the reduction of
Cu(II) to Cu(I) by APP involves an electron-transfer reaction and could
also lead to a production of hydroxyl radicals. Thus, copper
-mediated toxicity of APP-Cu(II)/(I) complexes may contribute to
neurodegeneration in AD.
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L21 ANSWER 2 OF 23 MEDLINE on STN
 ACCESSION NUMBER: 97417053 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9271002
 TITLE: Effects of cadmium, copper, and zinc and beta APP processing and turnover in COS-7 and PC12 cells. Relationship to Alzheimer disease pathology.
 AUTHOR: Smedman M; Potempska A; Rubenstein R; Ju W; Ramakrishna N; Denman R B
 CORPORATE SOURCE: New York State Institute for Basic Research in Developmental Disabilities, Staten Island 10314, USA.
 CONTRACT NUMBER: AGO 4220 (NIA) R29 25301
 SOURCE: Molecular and chemical neuropathology / sponsored by the International Society for Neurochemistry and the World Federation of Neurology and research groups on neurochemistry and cerebrospinal fluid, (1997 May) Vol. 31, No. 1, pp. 13-28. Journal code: 8910358. ISSN: 1044-7393.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199710
 ENTRY DATE: Entered STN: 24 Oct 1997
 Last Updated on STN: 3 Mar 2000
 Entered Medline: 10 Oct 1997

AB The effects of cadmium, copper, and zinc on beta APP metabolism were investigated in COS-7 and PC12 cells. Cadmium chloride (CdCl₂) increased beta APP steady-state protein levels and decreased beta APP posttranslational processing. These changes were not accompanied by alterations in beta APP mRNA levels or splicing. In addition, cytosolic alpha-actin and G3PDH levels were not affected. Further, neither zinc (ZnCl₂) nor copper (CuSO₄) altered beta APP levels or affected its normal processing. Pulse-chase studies revealed that the rate of beta APP maturation decreased twofold in the presence of 25 micromM CdCl₂ compared to untreated controls. beta APP secretion from the cell also dramatically slowed. These two factors result in the accumulation of partially processed beta APP inside cells. The presence of CdCl₂ also decreased the amount of an 8-kDa beta APP C-terminal fragment, indicating that the cellular compartment in which beta APP accumulates is not accessible to alpha-secretase. Studies using Brefeldin A suggest that this compartment may be the cis or medial Golgi. However, A beta production was proportionately increased. These data show that CdCl₂ can modulate the beta APP cleavage to favor A beta. Finally, beta APP mis-metabolism was shown to be unrelated to the hsp70 induction elicited by CdCl₂; both heat shock and CuSO₄ induced hsp70 but had no effect on steady-state levels of beta APP, although heat shock did slow beta APP maturation. These data indicate that hsp70 alone cannot chaperone beta APP through an alternate processing pathway leading to A beta production.

L21 ANSWER 3 OF 23 MEDLINE on STN
 ACCESSION NUMBER: 97324976 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9181045
 TITLE: Amyloid precursor protein, copper and Alzheimer's disease.
 AUTHOR: Multhaup G
 CORPORATE SOURCE: ZMBH Center for Molecular Biology, University of Heidelberg, Germany.
 SOURCE: Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie, (1997) Vol. 51, No. 3, pp. 105-11. Ref: 72

Journal code: 8213295. ISSN: 0753-3322.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 24 Jul 1997
Last Updated on STN: 6 Feb 1998
Entered Medline: 14 Jul 1997

AB Although a consensus that Alzheimer's disease (AD) is a single disease has not yet been reached, the involvement of the amyloid precursor protein (APP) and beta A4 (A beta) in the pathologic changes advances our understanding of the underlying molecular alterations. Increasing evidence implicates oxidative stress in the neurodegenerative process of AD. This hypothesis is based on the toxicity of beta A4 in cell cultures, and the findings that aggregation of beta A4 can be induced by metal-catalyzed oxidation and that free oxygen radicals might be involved in APP metabolism. Another neurological disorder, familial amyotrophic lateral sclerosis (FALS), supports our view that AD and FALS might be linked through a common mechanism. In FALS, SOD-Cu(I) complexes are affected by hydrogen peroxide and free radicals are produced. In AD, the reduction of Cu(II) to Cu(I) by APP involves an electron-transfer reaction and could also lead to a production of hydroxyl radicals. Thus, copper-mediated toxicity of APP-Cu(II)/(I) complexes may contribute to neurodegeneration in AD.

L21 ANSWER 4 OF 23 MEDLINE on STN
ACCESSION NUMBER: 97134514 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8984650
TITLE: Alzheimer's precursor protein and the use of bathocuproine for determining reduction of copper(II).
AUTHOR: Sayre L M
SOURCE: Science (New York, N.Y.), (1996 Dec 13) Vol. 274, No. 5294, pp. 1933-4.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Commentary
Letter
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 6 Feb 1998
Entered Medline: 8 Jan 1997

L21 ANSWER 5 OF 23 MEDLINE on STN
ACCESSION NUMBER: 96173947 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8596911
TITLE: The amyloid precursor protein of Alzheimer's disease in the reduction of copper(II) to copper(I).
AUTHOR: Multhaup G; Schlicksupp A; Hesse L; Behr D; Ruppert T; Masters C L; Beyreuther K
CORPORATE SOURCE: ZMBH-Center for Molecular Biology Heidelberg, University of Heidelberg, Germany.
SOURCE: Science (New York, N.Y.), (1996 Mar 8) Vol. 271, No. 5254, pp. 1406-9.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199604

ENTRY DATE: Entered STN: 24 Apr 1996
Last Updated on STN: 3 Mar 2000
Entered Medline: 17 Apr 1996

AB The transition metal ion copper(II) has a critical role in chronic neurologic diseases. The amyloid precursor protein (APP) of Alzheimer's disease or a synthetic peptide representing its copper-binding site reduced bound copper(II) to copper(I). This copper ion-mediated redox reaction led to disulfide bond formation in APP, which indicated that free sulfhydryl groups of APP were involved. Neither superoxide nor hydrogen peroxide had an effect on the kinetics of copper(II) reduction. The reduction of copper(II) to copper(I) by APP involves an electron-transfer reaction and could enhance the production of hydroxyl radicals, which could then attack nearby sites. Thus, copper-mediated toxicity may contribute to neurodegeneration in Alzheimer's disease.

L21 ANSWER 6 OF 23 MEDLINE on STN
ACCESSION NUMBER: 95327937 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7604268
TITLE: Zinc and Alzheimer's disease.
AUTHOR: Fitzgerald D J
SOURCE: Science (New York, N.Y.), (1995 Jun 30) Vol. 268, No. 5219, pp. 1920; author reply 1921-3.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Commentary
Letter
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 22 Aug 1995
Last Updated on STN: 11 Jan 2003
Entered Medline: 7 Aug 1995

L21 ANSWER 7 OF 23 MEDLINE on STN
ACCESSION NUMBER: 95294022 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7775475
TITLE: Proteolytic processing of Alzheimer's disease beta A4 amyloid precursor protein in human platelets.
AUTHOR: Li Q X; Evin G; Small D H; Multhaup G; Beyreuther K; Masters C L
CORPORATE SOURCE: Department of Pathology, University of Melbourne, Parkville, Victoria, Australia.
SOURCE: The Journal of biological chemistry, (1995 Jun 9) Vol. 270, No. 23, pp. 14140-7.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199507
ENTRY DATE: Entered STN: 20 Jul 1995
Last Updated on STN: 3 Mar 2000
Entered Medline: 10 Jul 1995

AB The processing of amyloid precursor protein (APP) and production of beta A4 amyloid are events likely to influence the development and progression of Alzheimer's disease, since beta A4 is the major constituent of amyloid deposited in this disorder. Our previous studies showed that human platelets contain full-length APP (APPFL) and are a suitable substrate to study normal APP processing. In the present study, we show that a 22-kDa beta A4-containing carboxyl-terminal fragment (22-CTF) of APP is present in unstimulated platelets. Both APPFL and 22-CTF are proteolytically

degraded when platelets are activated with thrombin, collagen, or calcium ionophore A23187. Complete cleavage of APPFL and 22-CTF require the presence of extracellular calcium. Following stimulation in the presence of calcium, a new CTF of 17 kDa is generated, and the NH2-terminal epitope of beta A4 amyloid is lost. Preincubation of platelets with the cell-permeable cysteine protease inhibitors calpeptin, (2S,3S)-trans-epoxysuccinyl-L-leucyl-amido-3-methylbutane ethyl ester (E64d), Na alpha-p-tosyl-L-lysine chloromethyl ketone, or calcium chelator EGTA before platelet stimulation inhibits the degradation of both APPFL and 22-CTF. Divalent metal ions including zinc, copper, and cobalt inhibit the degradation of APPFL and 22-CTF. This study suggests that a calcium-dependent neutral cysteine protease is involved in the proteolytic processing of an amyloidogenic species of APP in human platelets.

L21 ANSWER 8 OF 23 MEDLINE on STN
 ACCESSION NUMBER: 95126976 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7826392
 TITLE: Stabilization of secondary structure of Alzheimer beta-protein by aluminum(III) ions and D-Asp substitutions.
 AUTHOR: Vyas S B; Duffy L K
 CORPORATE SOURCE: Department of Chemistry and Biochemistry, University of Alaska Fairbanks, Fairbanks 99775.
 CONTRACT NUMBER: R15AG08978 (NIA)
 SOURCE: Biochemical and biophysical research communications, (1995 Jan 17) Vol. 206, No. 2, pp. 718-23.
 Journal code: 0372516. ISSN: 0006-291X.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199502
 ENTRY DATE: Entered STN: 23 Feb 1995
 Last Updated on STN: 3 Feb 1997
 Entered Medline: 16 Feb 1995

AB The CD spectra of the D-Asp substituted analogs of amyloid peptides, beta 6-25 and beta 1-40, showed a distinct blue-shift on Al3+ complexation. The influence of Al3+ coordination was most significant on the triply substituted beta 1-40 (D-Asp 1,7,23). This analog showed a reduction of the minima near 210nm and a simultaneous increase in the maxima near 200nm as compared to the native L-Asp beta 1-40. These observations suggest that Al3+ interaction with D-Asp induces the peptide backbone to increase its antiparallel beta-sheet character. D-Asp substitution and chelation by Al3+ lead to increased stability of higher molecular weight species of beta 1-40, and thereby could increase the toxicity of the Alzheimer amyloid protein.

L21 ANSWER 9 OF 23 MEDLINE on STN
 ACCESSION NUMBER: 94320627 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7913895
 TITLE: The beta A4 amyloid precursor protein binding to copper.
 AUTHOR: Hesse L; Beher D; Masters C L; Multhaup G
 CORPORATE SOURCE: Center for Molecular Biology Heidelberg, University Heidelberg, Germany.
 SOURCE: FEBS letters, (1994 Jul 25) Vol. 349, No. 1, pp. 109-16.
 Journal code: 0155157. ISSN: 0014-5793.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: (COMPARATIVE STUDY)

Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199409
ENTRY DATE: Entered STN: 9 Sep 1994
Last Updated on STN: 3 Feb 1997
Entered Medline: 1 Sep 1994

AB Previously it has been shown that the extracellular domain of transmembrane beta A4 amyloid precursor protein (APP) includes binding sites for zinc(II) and for molecules of the extracellular matrix such as collagen, laminin and the heparin sulfate chains of proteoglycans (HSPGs). Here we report that APP also binds copper ions. A copper type II binding site was located within residues 135-155 of the cysteine-rich domain of APP695 which is present in all eight APP splice isoforms known so far. The two essential histidines in the type II copper binding site of APP are conserved in the related protein APLP2. Copper(II) binding is shown to inhibit homophilic APP binding. The identification of a copper(II) binding site in APP suggests that APP and APLP2 may be involved in electron transfer and radical reactions.

L21 ANSWER 10 OF 23 MEDLINE on STN

ACCESSION NUMBER: 94216331 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8163520

TITLE: Modulation of A beta adhesiveness and secretase site cleavage by zinc.

AUTHOR: Bush A I; Pettingell W H Jr; Paradis M D; Tanzi R E
CORPORATE SOURCE: Laboratory of Genetics and Aging, Massachusetts General Hospital, Harvard Medical School, Boston 02129.

SOURCE: The Journal of biological chemistry, (1994 Apr 22)
Vol. 269, No. 16, pp. 12152-8.
Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199405

ENTRY DATE: Entered STN: 6 Jun 1994
Last Updated on STN: 3 Mar 2000
Entered Medline: 26 May 1994

AB Abnormalities of zinc homeostasis occur in Alzheimer's disease (AD), a dementia characterized by the aggregation of A beta in the brain, and in Down syndrome, a condition characterized by premature AD. We studied the binding of Zn²⁺ to a synthetic peptide representing residues 1-40 (A beta 1-40), as well as other domains of A beta. Two classes of Zn²⁺ binding were identified by ⁶⁵Zn²⁺ labeling: highly specific pH-dependent high affinity (K_a = 107 nM) binding, and lower affinity (K_a = 5.2 microm) binding. Gel filtration chromatography identified monomeric, dimeric, and polymeric A beta species. Zinc induced a marked loss of A beta solubility upon chromatographic analysis. This was attributed to precipitation onto the column glass, which contains aluminosilicate, and was confirmed by the observation of zinc-accelerated precipitation of A beta by kaolin, a hydrated aluminum silicate suspension. Zinc binding also increased A beta resistance to tryptic cleavage at the secretase site, indicating that a small (<3 microm) increase in brain Zn²⁺ concentration could significantly alter A beta metabolism. We propose that elevated brain interstitial zinc levels may increase A beta adhesiveness and interfere with A beta catabolism. Consequently, abnormalities of regional zinc concentrations in the brains of patients with AD or Down syndrome may contribute to A beta amyloidosis in these disorders.

L21 ANSWER 11 OF 23 MEDLINE on STN
ACCESSION NUMBER: 93367485 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8360682
TITLE: Aluminum, iron, and zinc ions promote aggregation of physiological concentrations of beta-amyloid peptide.
AUTHOR: Mantyh P W; Ghilardi J R; Rogers S; DeMaster E; Allen C J; Stimson E R; Maggio J E
CORPORATE SOURCE: Molecular Neurobiology Laboratory (151), Veteran's Administration Medical Center, Minneapolis, Minnesota 55417.
CONTRACT NUMBER: GM15904 (NIGMS)
NS22961 (NINDS)
NS23970 (NINDS)
SOURCE: Journal of neurochemistry, (1993 Sep) Vol. 61, No. 3, pp. 1171-4.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199309
ENTRY DATE: Entered STN: 15 Oct 1993
Last Updated on STN: 3 Feb 1997
Entered Medline: 27 Sep 1993

AB A major pathological feature of Alzheimer's disease (AD) is the presence of a high density of amyloid plaques in the brain tissue of patients. The plaques are predominantly composed of human beta-amyloid peptide beta A4, a 40-mer whose neurotoxicity is related to its aggregation. Certain metals have been proposed as risk factors for AD, but the mechanism by which the metals may exert their effects is unclear. Radioiodinated human beta A4 has been used to assess the effects of various metals on the aggregation of the peptide in dilute solution (10⁻¹⁰ M). In physiological buffers, 10⁻³ M calcium, cobalt, copper, manganese, magnesium, sodium, or potassium had no effect on the rate of beta A4 aggregation. In sharp contrast, aluminum, iron, and zinc under the same conditions strongly promoted aggregation (rate enhancement of 100-1,000-fold). The aggregation of beta A4 induced by aluminum and iron is distinguishable from that induced by zinc in terms of rate, extent, pH and temperature dependence. These results suggest that high concentrations of certain metals may play a role in the pathogenesis of AD by promoting aggregation of beta A4.

L21 ANSWER 12 OF 23 MEDLINE on STN
ACCESSION NUMBER: 89319583 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2473595
TITLE: The ultrastructural localization of sulfated proteoglycans is identical in the amyloids of Alzheimer's disease and AA, AL, senile cardiac and medullary carcinoma-associated amyloidosis.
AUTHOR: Young I D; Willmer J P; Kisilevsky R
CORPORATE SOURCE: Department of Pathology, Queen's University, Kingston, Ontario, Canada.
SOURCE: Acta neuropathologica, (1989) Vol. 78, No. 2, pp. 202-9.
Journal code: 0412041. ISSN: 0001-6322.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198908
ENTRY DATE: Entered STN: 9 Mar 1990

Last Updated on STN: 6 Feb 1998

Entered Medline: 21 Aug 1989

AB The cationic dyes cuproline blue and ruthenium red were used to ultrastructurally localize proteoglycans (PGs) within the neuritic plaque and neurofibrillary tangle of Alzheimer's disease. Highly sulfated PGs were specifically localized to the amyloid fibril of the neuritic plaque and the paired filaments of the neurofibrillary tangle. This demonstrates that highly sulfated PGs either comprise part of the Alzheimer's amyloid fibril and paired filament or are intimately associated with them. Four unrelated types of amyloid--AA (inflammation-associated), AL (immunoglobulin light chain), senile cardiac (prealbumin) and medullary carcinoma-associated amyloid (procalcitonin)--showed an identical pattern of localization of highly sulfated PG to the different amyloid fibrils. This constant close spatial relationship between PGs and diverse amyloid proteins suggests that PGs may play a role in amyloidogenesis.

L21 ANSWER 13 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:50722 BIOSIS

DOCUMENT NUMBER: PREV199800050722

TITLE: Effect of dietary zinc and copper on beta-amyloid precursor protein expression in the rat brain.

AUTHOR(S): Davis, Cindy D. [Reprint author]

CORPORATE SOURCE: USDA, ARS, GFHNRC, PO Box 9034, University Station, Grand Forks, ND 58202-9034, USA

SOURCE: Journal of Trace Elements in Experimental Medicine, (1997) Vol. 10, No. 4, pp. 249-258. print.
CODEN: JTEMEM. ISSN: 0896-548X.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 27 Jan 1998

Last Updated on STN: 27 Jan 1998

AB The beta-amyloid precursor protein (APP) is the source of the amyloid beta-peptide that accumulates in the brain in Alzheimer's disease. Recently, APP has been shown to bind zinc and copper, and this binding has been suggested to control APP conformation and stability. In vitro studies show that zinc ions cause beta-amyloid protein to form plaques resembling the amyloid plaques found in the brains of patients with Alzheimer's disease. This suggests a role for zinc and/or copper in the neuropathogenesis of Alzheimer's disease. Male Sprague-Dawley rats (100 +/- 10 were fed diets containing 5, 35, or 350 mug zinc/g diet, and 1.5, 3 or 6 mug copper/g diet for 6 weeks. Brain APP expression was determined by using Western blots. Proteins were separated on 8.5% SDS-PAGE, and the APP immunoreactive species were detected by using anti-Alzheimer precursor protein A4 clone 22C11. Alterations in dietary zinc and copper significantly ($P < 0.05$) affected ceruloplasmin, red blood cell and extracellular superoxide dismutase activities, and tissue mineral concentrations. Although brain zinc concentrations were 13% lower ($P < 0.005$) in animals fed low dietary zinc than in animals fed high dietary zinc, and brain copper concentrations were 11% lower ($P < 0.0001$) in animals fed low dietary copper than in animals fed high dietary copper, there were no significant differences in the expression of APP among the different dietary treatments. Therefore, it seems that dietary zinc and copper do not affect APP expression in the rat brain.

L21 ANSWER 14 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:530474 BIOSIS

DOCUMENT NUMBER: PREV199799829677

TITLE: Alzheimer A-beta peptides simultaneously reduce metals and produce reactive oxygen species.

AUTHOR(S): Huang, X. [Reprint author]; Atwood, C. S. [Reprint author];

Goldstein, L. E. [Reprint author]; Hartshorn, M. A. [Reprint author]; Moir, R. D.; Multhaup, G. [Reprint author]; Tanzi, R. E.; Bush, A. I. [Reprint author]
 CORPORATE SOURCE: Genet. and Aging Unit, Harv. Med. Sch., Mass. Gen. Hosp., Charlestown, MA 02129, USA
 SOURCE: Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 1663.
 Meeting Info.: 27th Annual Meeting of the Society for Neuroscience. New Orleans, Louisiana, USA. October 25-30, 1997.
 ISSN: 0190-5295.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 12 Dec 1997
 Last Updated on STN: 12 Dec 1997

L21 ANSWER 15 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:206954 BIOSIS
 DOCUMENT NUMBER: PREV199799506157
 TITLE: Neuroprotective action of cycloheximide involves induction of Bcl-2 and antioxidant pathways.
 AUTHOR(S): Furukawa, Katsutoshi [Reprint author]; Estus, Steven; Fu, Weiming; Mark, Robert J.; Mattson, Mark P.
 CORPORATE SOURCE: Sanders-Brown Res. Cent. Aging, Dep. Anatomy Neurobiol., Univ. Ky., 211 Sanders-Brown Build., 800 South Limestone, Lexington, KY 10537-0230, USA
 SOURCE: Journal of Cell Biology, (1997) Vol. 136, No. 5, pp. 1137-1149.
 CODEN: JCLBA3. ISSN: 0021-9525.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 12 May 1997
 Last Updated on STN: 12 May 1997

AB The ability of the protein synthesis inhibitor cycloheximide (CHX) to prevent neuronal death in different paradigms has been interpreted to indicate that the cell death process requires synthesis of "killer" proteins. On the other hand, data indicate that neurotrophic factors protect neurons in the same death paradigms by inducing expression of neuroprotective gene products. We now provide evidence that in embryonic rat hippocampal cell cultures, CHX protects neurons against oxidative insults by a mechanism involving induction of neuroprotective gene products including the antiapoptotic gene bcl-2 and antioxidant enzymes. Neuronal survival after exposure to glutamate, FeSO₄, and amyloid beta-peptide was increased in cultures pretreated with CHX at concentrations of 50-500 nM; higher and lower concentrations were ineffective. Neuroprotective concentrations of CHX caused only a moderate (20-40%) reduction in overall protein synthesis, and induced an increase in c-fos, c-jun, and bcl-2 mRNAs and protein levels as determined by reverse transcription-PCR analysis and immunocytochemistry, respectively. At neuroprotective CHX concentrations, levels of c-fos heteronuclear RNA increased in parallel with c-fos mRNA, indicating that CHX acts by inducing transcription. Neuroprotective concentrations of CHX suppressed accumulation of H₂O₂ induced by FeSO₄, suggesting activation of antioxidant pathways. Treatment of cultures with an antisense oligodeoxynucleotide directed against bcl-2 mRNA decreased Bcl-2 protein levels and significantly reduced the neuroprotective action of CHX, suggesting that induction of Bcl-2 expression was mechanistically involved in the neuroprotective actions of CHX. In addition, activity levels of the antioxidant enzymes Cu/Zn-superoxide dismutase, Mn-superoxide dismutase, and catalase were significantly increased in cultures exposed to neuroprotective levels of CHX. Our data suggest that low concentrations of CHX can promote neuron survival by inducing increased

levels of gene products that function in antioxidant pathways, a neuroprotective mechanism similar to that used by neurotrophic factors.

L21 ANSWER 16 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1996:551317 BIOSIS
DOCUMENT NUMBER: PREV199699273673
TITLE: Gene-targeted mice for APP and Cu-Zn SOD-1 for studying A-beta metabolism and amyloid-related neuropathologies.
AUTHOR(S): Howland, D.; Reaume, A.; Savage, M.; Flood, D.; Trusko, S.; Lin, Y. G.; Pinsker, L.; Lang, D.; Greenberg, B.; Siman, R.; Scott, R.
CORPORATE SOURCE: Cephalon, 145 Brandywine Pkwy, West Chester, PA 19380, USA
SOURCE: Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 1172.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience. Washington, D.C., USA. November 16-21, 1996.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1996
Last Updated on STN: 23 Jan 1997

L21 ANSWER 17 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1995:278158 BIOSIS
DOCUMENT NUMBER: PREV199598292458
TITLE: Function of amyloid beta-A4 protein and its precursor APP in health and disease.
AUTHOR(S): Beyreuther, Konrad [Reprint author]; Masters, Colin L.
CORPORATE SOURCE: ZMBH, Univ. Heidelberg, D-69120 Heidelberg, Germany
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0, No. 21B, pp. 87.
Meeting Info.: Keystone Symposium on the Molecular and Cellular Basis of Human Neurodegenerative Disease. Breckenridge, Colorado, USA. April 3-9, 1995.
ISSN: 0733-1959.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jul 1995
Last Updated on STN: 2 Aug 1995

L21 ANSWER 18 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1995245815 EMBASE
TITLE: Genetic approaches to pathogenesis of neurodegenerative diseases.
AUTHOR: Orr H.T.; Clark H.B.
CORPORATE SOURCE: H.T. Orr, Laboratory Medicine/Pathology Dept., Institute of Human Genetics, University of Minnesota, Minneapolis, MN, United States
SOURCE: Laboratory Investigation, (1995) Vol. 73, No. 2, pp. 161-171.
ISSN: 0023-6837 CODEN: LAINAW
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 022 Human Genetics
029 Clinical and Experimental Biochemistry
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English

ENTRY DATE: Entered STN: 12 Sep 1995
Last Updated on STN: 12 Sep 1995

L21 ANSWER 19 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1995214417 EMBASE
TITLE: Zinc and Alzheimer's disease.
AUTHOR: Fitzgerald D.J.; Maggio J.E.; Esler W.P.; Stimson E.R.;
Jennings J.M.; Ghilardi J.R.; Mantyh P.W.
CORPORATE SOURCE: D.J. Fitzgerald, Public Environmental Health Service, South
Australian Health Commission, Adelaide, SA 5000, Australia
SOURCE: Science, (1995) Vol. 268, No. 5219, pp. 1920-1923.
ISSN: 0036-8075 CODEN: SCIEAS
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Aug 1995
Last Updated on STN: 3 Aug 1995

L21 ANSWER 20 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1995196523 EMBASE
TITLE: Potent inhibitors of proteasome.
AUTHOR: Iqbal M.; Chatterjee S.; Kauer J.C.; Das M.; Messina P.;
Freed B.; Biazzo W.; Siman R.
CORPORATE SOURCE: M. Iqbal, Dept. of Chemistry and Biochemistry, Cephalon,
Inc., 145 Brandywine Parkway, West Chester, PA 19380,
United States
SOURCE: Journal of Medicinal Chemistry, (1995) Vol. 38, No. 13, pp.
2276-2277.
ISSN: 0022-2623 CODEN: JMCMAR
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jul 1995
Last Updated on STN: 27 Jul 1995

L21 ANSWER 21 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1993202849 EMBASE
TITLE: Corpora amylacea could be an indicator of
neurodegeneration.
AUTHOR: Singhrao S.K.; Neal J.W.; Newman G.R.
CORPORATE SOURCE: Mrs. S.K. Singhrao, EM Unit, Univ of Wales College of
Medicine, Heath Park, Cardiff, United Kingdom
SOURCE: Neuropathology and Applied Neurobiology, (1993) Vol. 19,
No. 3, pp. 269-276.
ISSN: 0305-1846 CODEN: NANEDL
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 Aug 1993
Last Updated on STN: 8 Aug 1993
AB We describe an investigation of corpora amylacea (CA) in the brain tissue
of Alzheimer's disease (AD) cases and normal ageing controls, using both

light (LM) and electron (EM) microscopic techniques. CA populations were shown by routine histological staining of LR White resin sections with methenamine silver and PAS, and were compared with those shown by immunocytochemistry using antibodies to tau, GFAP, tubulin, ubiquitin, β -amyloid and serum amyloid P component in serial sections. All CA were immunoreactive with anti-tau and all were unreactive with anti- β -amyloid. Most were immunoreactive with anti-serum amyloid P component, although this was often weak in AD. CA from normal ageing brain were immunoreactive for proteins that are associated with the neuronal cytoskeleton and cell injury. CA from AD brain shared some of these but differed from those in normal ageing brain by being in much larger number and more variable in their immunoreactivity. In all CA, X-ray microanalysis illustrated the presence of the metallic elements Ca, Fe and Cu. Aluminium, often associated with AD, was not present, even in CA from AD brain. Phosphorus and sulphur, probably from phosphorylated proteins associated with degenerating cytoskeleton elements, were usually detected. In AD brain, the greater numbers of CA and their variable biochemical and elemental composition, when compared with CA in the normal ageing brain, suggests that they may derive from a number of sources both neuronal and glial as a result of the neurodegenerative disease.

L21 ANSWER 22 OF 23 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1996:341056 SCISEARCH
 THE GENUINE ARTICLE: UH594
 TITLE: Zinc and Alzheimer's disease
 AUTHOR: Nachev P C (Reprint); Larner A J
 CORPORATE SOURCE: UNIV CAMBRIDGE, DEPT ANAT, CAMBRIDGE CB2 3DY, ENGLAND;
 UNIV CAMBRIDGE, ADDENBROOKES HOSP, DEPT NEUROL, CAMBRIDGE, ENGLAND
 COUNTRY OF AUTHOR: ENGLAND
 SOURCE: TRACE ELEMENTS AND ELECTROLYTES, (1996) Vol. 13,
 No. 2, pp. 55-59.
 ISSN: 0174-7371.
 PUBLISHER: DUSTRI-VERLAG DR KARL FEISTLE, BAHNHOFSTRABE 9 POSTFACH
 49, W-8024 MUNCHEN-DEISENHOFEN, GERMANY.
 DOCUMENT TYPE: General Review; Journal
 FILE SEGMENT: CLIN
 LANGUAGE: English
 REFERENCE COUNT: 45
 ENTRY DATE: Entered STN: 1996
 Last Updated on STN: 1996

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The evidence that zinc may play a role in the pathogenesis of Alzheimer's disease is reviewed. It has been suggested that cerebral zinc metabolism could be involved in the pathogenesis of both the principal neuropathological hallmarks of Alzheimer's disease: excess zinc may facilitate aggregation and deposition of amyloid, whereas zinc deficiency may hasten neurofibrillary tangle formation. Studies of zinc levels within the tissues (blood, cerebrospinal fluid, brain) of patients with Alzheimer's disease have produced variable results which have not resolved these issues. Hence, at our current level of understanding, treatment of Alzheimer's disease with either zinc supplementation or chelation cannot be generally advocated. However, zinc supplementation in patients with severe cognitive impairment and evidence of zinc deficiency would be justifiable.

L21 ANSWER 23 OF 23 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1993:471131 SCISEARCH
 THE GENUINE ARTICLE: LN830
 TITLE: OXIDATIVE STRESS IN SOME DEMENTIA TYPES
 AUTHOR: ROCHE E (Reprint); ROMEROALVIRA D
 CORPORATE SOURCE: HOSP INSALUD, SERV CARDIOL, ZARAGOZA, SPAIN; CTR MED UNIV

COUNTRY OF AUTHOR: GENEVA, DEPT BIOCHIM CLIN, CH-1211 GENEVA 4, SWITZERLAND
SOURCE: SPAIN; SWITZERLAND
MEDICAL HYPOTHESES, (JUN 1993) Vol. 40, No. 6,
pp. 342-350.
ISSN: 0306-9877.
PUBLISHER: CHURCHILL LIVINGSTONE, JOURNAL PRODUCTION DEPT, ROBERT
STEVENSON HOUSE, 1-3 BAXTERS PLACE, LEITH WALK, EDINBURGH,
MIDLOTHIAN, SCOTLAND EH1 3AF.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 72
ENTRY DATE: Entered STN: 1994
Last Updated on STN: 1994

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB By analogy to some pathologies (such as demyelinating diseases, arthritis and inflammatory processes) where the loss of cellular integrity is the starting point of tissue oxidative damage, it is proposed that some dementia types could be derived from a similar mechanism. The following oxidative events are proposed: (a) different agents could alter capillary or neuron integrity with the subsequent leakage of oxidases, proteases and transition metals from cellular compartments; (b) the persistence of the damaging agent, possible depletion of antioxidative defenses and concomitant loss of neuron function; (c) alteration of adjacent cells in the same manner; and (d) finally localized brain necrosis and progression of the dementia.

=>

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FILE COVERS 1907 - 17 Oct 2007 VOL 147 ISS 17
FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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=> E BUSH ASHLEY I/IN 25

E1	1	BUSH ARTHUR B JR/IN
E2	1	BUSH ASHLEY/IN
E3	10 -->	BUSH ASHLEY I/IN
E4	3	BUSH ASHLEY IAN/IN
E5	1	BUSH ASHLEY L/IN
E6	1	BUSH BRADLEY S/IN
E7	1	BUSH BRADLEY STEPHEN/IN
E8	1	BUSH BRIAN/IN
E9	2	BUSH BRIAN DAVID/IN
E10	1	BUSH C ALLEN/IN
E11	1	BUSH CAROL L/IN
E12	1	BUSH CAROLE/IN
E13	1	BUSH CATHERINE/IN
E14	1	BUSH CHARLENE ELEANOR/IN
E15	1	BUSH CHARLES HUNTER/IN
E16	1	BUSH CHARLES L JR/IN
E17	8	BUSH CHARLES N/IN
E18	2	BUSH CHARLES NEAL/IN
E19	1	BUSH CHRISTOPHER/IN
E20	2	BUSH CHRISTOPHER DAVID/IN
E21	1	BUSH CHRISTOPHER JOHN THOMSON/IN
E22	1	BUSH CHRISTOPHER N/IN
E23	3	BUSH CONDON S/IN
E24	1	BUSH CRAIG P/IN
E25	2	BUSH CRAIG PALMER/IN

=> S (E3) AND (REDOX AMYLOID)

10 "BUSH ASHLEY I"/IN
140874 REDOX
8 REDOXES
140877 REDOX
(REDOX OR REDOXES)
28164 AMYLOID
1756 AMYLOIDS
28263 AMYLOID

(AMYLOID OR AMYLOIDS)
0 REDOX AMYLOID

(REDOX (W) AMYLOID)

L1 0 ("BUSH ASHLEY I"/IN) AND (REDOX AMYLOID)


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=> S (E3) AND (REDOX )
      10 "BUSH ASHLEY I"/IN
      140874 REDOX
      8 REDOXES
      140877 REDOX
        (REDOX OR REDOXES)
L2      4 ("BUSH ASHLEY I"/IN) AND (REDOX )

=> S (E3) AND (REDOX, CROSSLINKING)
      10 "BUSH ASHLEY I"/IN
      140874 REDOX
      8 REDOXES
      140877 REDOX
        (REDOX OR REDOXES)
      206119 CROSSLINKING
      137 CROSSLINKINGS
      206180 CROSSLINKING
        (CROSSLINKING OR CROSSLINKINGS)
      25 REDOX, CROSSLINKING
        (REDOX (W) CROSSLINKING)
L3      0 ("BUSH ASHLEY I"/IN) AND (REDOX, CROSSLINKING)

=> S (E3) AND (REDOX REACTIVE METAL)
      10 "BUSH ASHLEY I"/IN
      140874 REDOX
      8 REDOXES
      140877 REDOX
        (REDOX OR REDOXES)
      320465 REACTIVE
      160 REACTIVES
      320576 REACTIVE
        (REACTIVE OR REACTIVES)
      1780448 METAL
      888866 METALS
      2153783 METAL
        (METAL OR METALS)
      6 REDOX REACTIVE METAL
        (REDOX (W) REACTIVE (W) METAL)
L4      0 ("BUSH ASHLEY I"/IN) AND (REDOX REACTIVE METAL)

=> S (E3) AND (REDOX )
      10 "BUSH ASHLEY I"/IN
      140874 REDOX
      8 REDOXES
      140877 REDOX
        (REDOX OR REDOXES)
L5      4 ("BUSH ASHLEY I"/IN) AND (REDOX )

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=> DIS L5 1 IBIB IABS
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L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2005:453721 CAPLUS
 DOCUMENT NUMBER: 142:476270
 TITLE: Method of screening for drugs useful in treating
 Alzheimer's disease based on alteration of production of
 reduced metal ions and hydrogen peroxide
 INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood,
 Craig S.; Tanzi, Rudolph E.
 PATENT ASSIGNEE(S): The General Hospital Corporation, USA
 SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S.
 Ser. No. 380,704.

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005112543	A1	20050526	US 2003-643226	20030819
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
PRIORITY APPLN. INFO.:				
			WO 1998-US4683	W 19980311
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606
			US 1997-816122	A2 19970311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908

ABSTRACT:

The invention discloses methods for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions. The methodol. of the invention involves determining whether the agent is capable of altering production of hydrogen peroxide, Cu(I), or Fe(II).

=> DIS L5 2 IBIB IABS

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L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:157611 CAPLUS

DOCUMENT NUMBER: 136:194219

TITLE: Method for the identification of agents that inhibit or promote cataracts and uses thereof

INVENTOR(S): Bush, Ashley I.; Goldstein, Lee E.

PATENT ASSIGNEE(S): The General Hospital Corporation, USA

SOURCE: PCT Int. Appl., 64 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002015942	A1	20020228	WO 2000-US25975	20000922
WO 2002015942	A8	20030213		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,				

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
 CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2452346	A1	20020228	CA 2000-2452346	20000922
AU 200076021	A	20020304	AU 2000-76021	20000922
EP 1311299	A1	20030521	EP 2000-965284	20000922
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2004506915	T	20040304	JP 2002-520863	20000922
US 2005084918	A1	20050421	US 2004-983780	20041109
US 7166472	B2	20070123		

PRIORITY APPLN. INFO.: US 2000-226125P P 20000818
 WO 2000-US25975 W 20000922
 US 2003-344860 A3 20030718

ABSTRACT:

Described are methods for the identification of agents useful in the treatment or prevention of cataracts. Also described are methods for the identification of agents that may inadvertently promote or accelerate the formation of cataracts, and methods of treating or preventing injuries to or diseases of the ocular lens, retina and/or macula. More specifically, the invention describes methods for the identification of pharmacol. agents useful in treating cataracts by inhibiting the crosslinking of eye lens proteins.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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CA SUBSCRIBER PRICE	-1.56	-1.56

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	35.95
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-1.56

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FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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L5 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:790364 CAPLUS
DOCUMENT NUMBER: 133:344631
TITLE: Method of screening for drugs useful in treating
Alzheimer's disease
INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood,
Craig S.; Tanzi, Rudolph E.
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066181	A1	20001109	WO 2000-US11715	20000501
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,				
CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,				
ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,				
LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,				
SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,				
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,				
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2371768	A1	20001109	CA 2000-2371768	20000501
EP 1196198	A1	20020417	EP 2000-928644	20000501
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				
IE, SI, LT, LV, FI, RO				
JP 2002543402	T	20021217	JP 2000-615064	20000501
AU 776951	B2	20040930	AU 2000-46849	20000501
PRIORITY APPLN. INFO.:			US 1999-131579P	P 19990429
			WO 2000-US11715	W 20000501

ABSTRACT:
Methods are provided for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	3.30	39.25
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL

	ENTRY	SESSION
CA SUBSCRIBER PRICE	-0.78	-2.34

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	ENTRY	SESSION
FULL ESTIMATED COST	0.06	39.31
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-2.34

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FILE COVERS 1907 - 17 Oct 2007 VOL 147 ISS 17
 FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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=> DIS L5 4 IBIB IABS
 THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
 DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L5 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1998:621114 CAPLUS
 DOCUMENT NUMBER: 129:239902
 TITLE: Identification of agents for use in the treatment of Alzheimer's disease, and methods and compositions for treatment of conditions caused by amyloidosis and/or A β -mediated ROS formation
 INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.; Tanzi, Rudolph E.
 PATENT ASSIGNEE(S): The General Hospital Corp., USA
 SOURCE: PCT Int. Appl., 198 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2284170	A1	19980917	CA 1998-2284170	19980311
AU 9865484	A	19980929	AU 1998-65484	19980311
AU 748768	B2	20020613		
EP 1007048	A1	20000614	EP 1998-911551	19980311
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001514661	T	20010911	JP 1998-539718	19980311
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
AU 2002301084	A1	20030227	AU 2002-301084	20020912
US 2005112543	A1	20050526	US 2003-643226	20030819
PRIORITY APPLN. INFO.:			US 1997-816122	A2 19970311
			AU 1998-65484	A3 19980311
			WO 1998-US4683	W 19980311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606

ABSTRACT:

The invention relates to the identification of pharmacol. agents to be used in the treatment of Alzheimer's disease and related pathol. conditions. Methods and compns. for treatment of conditions caused by amyloidosis, A β -mediated ROS formation, or both, such as Alzheimer's disease, are disclosed.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE
COST IN U.S. DOLLARS

SINCE FILE ENTRY	TOTAL SESSION
3.30	42.61

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE ENTRY	TOTAL SESSION
-0.78	-3.12

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Oct 12, 2007 (20071012/UP).

=> FIL CAPLUS
COST IN U.S. DOLLARS

SINCE FILE ENTRY	TOTAL SESSION
0.06	42.67

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE ENTRY	TOTAL SESSION
0.00	-3.12

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FILE COVERS 1907 - 17 Oct 2007 VOL 147 ISS 17
FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

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=> E HUANG XUDONG/IN 25

E1	1	HUANG XUANYI/IN
E2	1	HUANG XUBIN/IN
E3	17 -->	HUANG XUDONG/IN
E4	3	HUANG XUE F/IN
E5	1	HUANG XUE LIN/IN
E6	2	HUANG XUEBIN/IN
E7	4	HUANG XUECHAO/IN
E8	2	HUANG XUEFEI/IN
E9	1	HUANG XUEFEN/IN
E10	2	HUANG XUEFENG/IN
E11	3	HUANG XUEHONG/IN
E12	1	HUANG XUEHUA/IN
E13	5	HUANG XUEHUI/IN
E14	36	HUANG XUEJIE/IN
E15	2	HUANG XUEJIN/IN
E16	1	HUANG XUEJING/IN
E17	1	HUANG XUEJU/IN
E18	1	HUANG XUEJUE/IN
E19	3	HUANG XUEJUN/IN
E20	1	HUANG XUELIANG/IN
E21	2	HUANG XUELUN/IN
E22	3	HUANG XUEMEI/IN
E23	5	HUANG XUEMIN/IN
E24	1	HUANG XUENAN/IN
E25	1	HUANG XUENGUANG/IN

=> S (E3) AND (REDOX)

17 "HUANG XUDONG"/IN
140874 REDOX
8 REDOXES
140877 REDOX
(REDOX OR REDOXES)

L6 3 ("HUANG XUDONG"/IN) AND (REDOX)

=> DIS L6 1 IBIB IABS

THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L6 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:453721 CAPLUS
 DOCUMENT NUMBER: 142:476270
 TITLE: Method of screening for drugs useful in treating
 Alzheimer's disease based on alteration of production of
 reduced metal ions and hydrogen peroxide
 INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood,
 Craig S.; Tanzi, Rudolph E.
 PATENT ASSIGNEE(S): The General Hospital Corporation, USA
 SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S.
 Ser. No. 380,704.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005112543	A1	20050526	US 2003-643226	20030819
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
PRIORITY APPLN. INFO.:			WO 1998-US4683	W 19980311
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606
			US 1997-816122	A2 19970311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908

ABSTRACT:

The invention discloses methods for identifying candidate pharmacol. agents to
 be used in the treatment and/or prevention of Alzheimer's disease and/or
 related pathol. conditions. The methodol. of the invention involves determining
 whether the agent is capable of altering production of hydrogen peroxide, Cu(I), or
 Fe(II).

=> FIL STNGUIDE
 COST IN U.S. DOLLARS
 FULL ESTIMATED COST

SINCE FILE	TOTAL
ENTRY	SESSION
7.32	49.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
 CA SUBSCRIBER PRICE

SINCE FILE	TOTAL
ENTRY	SESSION
-0.78	-3.90

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 COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION

FULL ESTIMATED COST	1.26	51.25
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-3.90

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 FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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=> DIS L6 2 IBIB IABS
 THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
 DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L6 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2000:790364 CAPLUS
 DOCUMENT NUMBER: 133:344631
 TITLE: Method of screening for drugs useful in treating Alzheimer's disease
 INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.; Tanzi, Rudolph E.
 PATENT ASSIGNEE(S): The General Hospital Corporation, USA
 SOURCE: PCT Int. Appl., 98 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066181	A1	20001109	WO 2000-US11715	20000501
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
CA 2371768	A1	20001109	CA 2000-2371768	20000501
EP 1196198	A1	20020417	EP 2000-928644	20000501
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			

JP 2002543402	T	20021217	JP 2000-615064	20000501
AU 776951	B2	20040930	AU 2000-46849	20000501
PRIORITY APPLN. INFO.:			US 1999-131579P	P 19990429
			WO 2000-US11715	W 20000501

ABSTRACT:

Methods are provided for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
3.30	54.55

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-0.78	-4.68

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=> FIL CAPLUS
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.06	54.61

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
0.00	-4.68

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FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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=> DIS L6 3 IBIB IABS
THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS

DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L6 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1998:621114 CAPLUS

DOCUMENT NUMBER: 129:239902

TITLE: Identification of agents for use in the treatment of Alzheimer's disease, and methods and compositions for treatment of conditions caused by amyloidosis and/or A β -mediated ROS formation

INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.; Tanzi, Rudolph E.

PATENT ASSIGNEE(S): The General Hospital Corp., USA

SOURCE: PCT Int. Appl., 198 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			
CA 2284170	A1	19980917	CA 1998-2284170	19980311
AU 9865484	A	19980929	AU 1998-65484	19980311
AU 748768	B2	20020613		
EP 1007048	A1	20000614	EP 1998-911551	19980311
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
JP 2001514661	T	20010911	JP 1998-539718	19980311
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
AU 2002301084	A1	20030227	AU 2002-301084	20020912
US 2005112543	A1	20050526	US 2003-643226	20030819
PRIORITY APPLN. INFO.:			US 1997-816122	A2 19970311
			AU 1998-65484	A3 19980311
			WO 1998-US4683	W 19980311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606

ABSTRACT:

The invention relates to the identification of pharmacol. agents to be used in the treatment of Alzheimer's disease and related pathol. conditions. Methods and compns. for treatment of conditions caused by amyloidosis, A β -mediated ROS formation, or both, such as Alzheimer's disease, are disclosed.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

3.30

57.91

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

CA SUBSCRIBER PRICE	ENTRY -0.78	SESSION -5.46
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FILE 'STNGUIDE' ENTERED AT 12:22:28 ON 17 OCT 2007
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FILE CONTAINS CURRENT INFORMATION.
 LAST RELOADED: Oct 12, 2007 (20071012/UP).

=> FIL CAPLUS		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.06	57.97
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-5.46

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FILE COVERS 1907 - 17 Oct 2007 VOL 147 ISS 17
 FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

<http://www.cas.org/infopolicy.html>

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=> E ATWOOD CRAIG S/IN 25
E1      2      ATWOOD BRYAN/IN
E2      1      ATWOOD CHARLES T/IN
E3      5 --> ATWOOD CRAIG S/IN
E4      2      ATWOOD DAN/IN
E5      2      ATWOOD DAVID A/IN
E6      3      ATWOOD DAVID ALLAN/IN
E7      3      ATWOOD DONALD K/IN
E8      1      ATWOOD E H/IN
E9      1      ATWOOD EDWARD N/IN
E10     2      ATWOOD EDWARDS S/IN
E11     3      ATWOOD EDWIN H/IN
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E13     3      ATWOOD EUGENE R/IN
E14     3      ATWOOD F C/IN
E15     31     ATWOOD FRANCIS C/IN
E16     3      ATWOOD FRANCIS CLARKE/IN
E17     7      ATWOOD GEO E/IN
E18     7      ATWOOD GEORGE E/IN
E19     3      ATWOOD GEORGE F/IN
E20     3      ATWOOD GILBERT R/IN
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E21 7 ATWOOD GILBERT RICHARD/IN
 E22 2 ATWOOD GLENN A/IN
 E23 4 ATWOOD GREG/IN
 E24 1 ATWOOD GREGORY/IN
 E25 3 ATWOOD GREGORY E/IN

=> S (E3) AND (REDOX)

5 "ATWOOD CRAIG S"/IN

140874 REDOX

8 REDOXES

140877 REDOX

(REDOX OR REDOXES)

L7 3 ("ATWOOD CRAIG S"/IN) AND (REDOX)

=> DIS L7 1 IBIB IABS

THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS

DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L7 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:453721 CAPLUS

DOCUMENT NUMBER: 142:476270

TITLE: Method of screening for drugs useful in treating
 Alzheimer's disease based on alteration of production of
 reduced metal ions and hydrogen peroxide

INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig
 S.; Tanzi, Rudolph E.

PATENT ASSIGNEE(S): The General Hospital Corporation, USA

SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S.
 Ser. No. 380,704.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005112543	A1	20050526	US 2003-643226	20030819
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
PRIORITY APPLN. INFO.:			WO 1998-US4683	W 19980311
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606
			US 1997-816122	A2 19970311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908

ABSTRACT:

The invention discloses methods for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions. The methodol. of the invention involves determining whether the agent is capable of altering production of hydrogen peroxide, Cu(I), or Fe(II).

=> DIS L7 2 IBIB IABS

THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L7 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:790364 CAPLUS
DOCUMENT NUMBER: 133:344631
TITLE: Method of screening for drugs useful in treating
Alzheimer's disease
INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig
S.; Tanzi, Rudolph E.
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066181	A1	20001109	WO 2000-US11715	20000501
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,				
CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,				
ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,				
LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,				
SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW,				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,				
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,				
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2371768	A1	20001109	CA 2000-2371768	20000501
EP 1196198	A1	20020417	EP 2000-928644	20000501
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				
IE, SI, LT, LV, FI, RO				
JP 2002543402	T	20021217	JP 2000-615064	20000501
AU 776951	B2	20040930	AU 2000-46849	20000501
PRIORITY APPLN. INFO.:			US 1999-131579P	P 19990429
			WO 2000-US11715	W 20000501

ABSTRACT:

Methods are provided for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
10.62	68.59

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-1.56	-7.02

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LAST RELOADED: Oct 12, 2007 (20071012/UP).

=> FIL CAPLUS

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	68.65
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-7.02

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FILE COVERS 1907 - 17 Oct 2007 VOL 147 ISS 17
 FILE LAST UPDATED: 16 Oct 2007 (20071016/ED)

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=> DIS L7 3 IBIB IABS
 THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
 DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L7 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1998:621114 CAPLUS
 DOCUMENT NUMBER: 129:239902
 TITLE: Identification of agents for use in the treatment of Alzheimer's disease, and methods and compositions for treatment of conditions caused by amyloidosis and/or A β -mediated ROS formation
 INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.; Tanzi, Rudolph E.
 PATENT ASSIGNEE(S): The General Hospital Corp., USA
 SOURCE: PCT Int. Appl., 198 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 9840071	A1	19980917	WO 1998-US4683	19980311
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			

CA 2284170	A1	19980917	CA 1998-2284170	19980311
AU 9865484	A	19980929	AU 1998-65484	19980311
AU 748768	B2	20020613		
EP 1007048	A1	20000614	EP 1998-911551	19980311
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001514661	T	20010911	JP 1998-539718	19980311
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
AU 2002301084	A1	20030227	AU 2002-301084	20020912
US 2005112543	A1	20050526	US 2003-643226	20030819
PRIORITY APPLN. INFO.:			US 1997-816122	A2 19970311
			AU 1998-65484	A3 19980311
			WO 1998-US4683	W 19980311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606

ABSTRACT:

The invention relates to the identification of pharmacol. agents to be used in the treatment of Alzheimer's disease and related pathol. conditions. Methods and compns. for treatment of conditions caused by amyloidosis, A β -mediated ROS formation, or both, such as Alzheimer's disease, are disclosed.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> E TANZI RUDOLPH E/IN 25

E1	1	TANZI MARIO/IN
E2	3	TANZI RUDOLPH/IN
E3	28 -->	TANZI RUDOLPH E/IN
E4	1	TANZI STEVEN/IN
E5	1	TANZIAWA TUNEYUKI/IN
E6	2	TANZILLI JAMES D/IN
E7	3	TANZILLI RICHARD A/IN
E8	1	TANZILLI RICHARD ANTHONY/IN
E9	1	TANZINI SAURO/IN
E10	2	TANZLER RICHARD/IN
E11	8	TANZMAN DANIEL P/IN
E12	1	TANZMAN HERBERT D/IN
E13	1	TANZMANN DANIEL/IN
E14	1	TANZMANN LUBOMIR CS/IN
E15	1	TANZMANN WOLFGANG/IN
E16	1	TANZMEIER PETER/IN
E17	4	TANZO ATSUHARU/IN
E18	11	TANZO JUNJI/IN
E19	1	TANZO TOMOHARU/IN
E20	4	TANZO TOMOJI/IN
E21	1	TANZOLA JOHN C/IN
E22	3	TANZOLA WM A/IN
E23	5	TANZOSH JAMES M/IN
E24	7	TANZYBAEVA L V/IN
E25	1	TANZYBAEVA LYUDMILA V/IN

=> S (E3) AND (REDOX)

28 "TANZI RUDOLPH E"/IN
140874 REDOX
8 REDOXES
140877 REDOX

(REDOX OR REDOXES)

L8 3 ("TANZI RUDOLPH E"/IN) AND (REDOX)

=> DIS L8 1 IBIB IABS

THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:453721 CAPLUS
DOCUMENT NUMBER: 142:476270
TITLE: Method of screening for drugs useful in treating
Alzheimer's disease based on alteration of production of
reduced metal ions and hydrogen peroxide
INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.;
Tanzi, Rudolph E.
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S.
Ser. No. 380,704.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005112543	A1	20050526	US 2003-643226	20030819
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
PRIORITY APPLN. INFO.:			WO 1998-US4683	W 19980311
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606
			US 1997-816122	A2 19970311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908

ABSTRACT:

The invention discloses methods for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions. The methodol. of the invention involves determining whether the agent is capable of altering production of hydrogen peroxide, Cu(I), or Fe(II).

=> DIS L8 2 IBIB IABS

THE ESTIMATED COST FOR THIS REQUEST IS 2.83 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L8 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:790364 CAPLUS
DOCUMENT NUMBER: 133:344631
TITLE: Method of screening for drugs useful in treating
Alzheimer's disease
INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.;
Tanzi, Rudolph E.
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066181	A1	20001109	WO 2000-US11715	20000501
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
CA 2371768	A1	20001109	CA 2000-2371768	20000501
EP 1196198	A1	20020417	EP 2000-928644	20000501
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			
JP 2002543402	T	20021217	JP 2000-615064	20000501
AU 776951	B2	20040930	AU 2000-46849	20000501
PRIORITY APPLN. INFO.:			US 1999-131579P	P 19990429
			WO 2000-US11715	W 20000501

ABSTRACT:

Methods are provided for identifying candidate pharmacol. agents to be used in the treatment and/or prevention of Alzheimer's disease and/or related pathol. conditions.

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=> DIS L8 3 IBIB IABS
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L8 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:621114 CAPLUS
DOCUMENT NUMBER: 129:239902
TITLE: Identification of agents for use in the treatment of Alzheimer's disease, and methods and compositions for treatment of conditions caused by amyloidosis and/or A β -mediated ROS formation
INVENTOR(S): Bush, Ashley I.; Huang, Xudong; Atwood, Craig S.; Tanzi, Rudolph E.
PATENT ASSIGNEE(S): The General Hospital Corp., USA
SOURCE: PCT Int. Appl., 198 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840071	A1	19980917	WO 1998-US4683	19980311
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			

CA 2284170	A1	19980917	CA 1998-2284170	19980311
AU 9865484	A	19980929	AU 1998-65484	19980311
AU 748768	B2	20020613		
EP 1007048	A1	20000614	EP 1998-911551	19980311
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001514661	T	20010911	JP 1998-539718	19980311
US 6638711	B1	20031028	US 2000-560883	20000428
US 7045531	B1	20060516	US 2000-380704	20000606
AU 2002301084	A1	20030227	AU 2002-301084	20020912
US 2005112543	A1	20050526	US 2003-643226	20030819
PRIORITY APPLN. INFO.:			US 1997-816122	A2 19970311
			AU 1998-65484	A3 19980311
			WO 1998-US4683	W 19980311
			US 1999-131579P	P 19990429
			US 1999-380704	A2 19990908
			US 2000-560883	A3 20000428
			US 2000-380704	A2 20000606

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The invention relates to the identification of pharmacol. agents to be used in the treatment of Alzheimer's disease, and related pathol. conditions. Methods and compns. for treatment of conditions caused by amyloidosis, A β -mediated ROS formation, or both, such as Alzheimer's disease, are disclosed.

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